clinical presentation of the patients, response to trivalent botulinum antitoxin, and isolation of toxigenic *C. butyricum* from one of the consumed food articles strongly suggest that the outbreak was caused by food contaminated with toxigenic *C. butyricum*.

Neurotoxigenic *C. butyricum* was first reported in 1986 in two cases of infant botulism in Rome (6). Recently, neurotoxigenic *C. butyricum* was isolated from the food implicated in an outbreak of clinically diagnosed type E botulism in China (7). In this outbreak, it appears that sevu, because of improper storage, was contaminated with the spores of *C. butyricum*, which subsequently germinated and produced toxin. To the best of our knowledge, this is the first report of neurotoxigenic *C. butyricum* causing foodborne botulism in India.

The changing epidemiology of foodborne disease as highlighted in this report calls for improved surveillance, including the development of new technology for identifying outbreaks.

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Molecular Analysis of *Salmonella paratyphi* A From an Outbreak in New Delhi, India

To the Editor: In the context of emerging infectious diseases, enteric fever caused by *Salmonella paratyphi* A deserves increased attention and vigilance, although its severity is often milder than that of *S. typhi* disease. Outbreaks associated with this organism are exceedingly rare but have recently been reported in India (1) and Thailand. In India, the first reported outbreak of disease associated with *S. paratyphi* A (1) provided an opportunity to study the molecular epidemiology of infection caused by this organism.

A total of 18 human blood isolates of *S. paratyphi* A, 13 from the outbreak in New Delhi, India (from September to October 1996) (1) and 5 sporadic isolates from cases unrelated to the outbreak, were used in this study. A total of 36 culture-positive cases were detected during the 6-week outbreak. All strains were phage type 1 and were sensitive to all antibiotics tested. Isolates were analyzed by ribotyping and pulsed-field gel electrophoresis (PFGE) (2,3). PFGE/ribotype profiles were assigned arbitrary designations and analyzed by defining a similarity (Dice) coefficient, F (3), where F = 1.0 indicates complete pattern identity and F = 0, complete dissimilarity.

The five sporadic isolates of *S. paratyphi* A gave PFGE patterns following *Xba*I (5'-TCTAGA-3') digestion that were unique and distinctly different, with differences of 8 to 12 bands (F = 0.63-0.70). In contrast, the 13 outbreak isolates shared only four closely related PFGE patterns differing only in 1 to 6 bands (F = 0.8-1.0). Among the outbreak strains, two distinct clones were

observed, X1 and X2, which differed by 5 to 6 bands. Furthermore, outbreak isolates X3 and X4 were closely related to X1, differing by four and three DNA fragments, respectively. Similar results were obtained after digestion with a second restriction endonuclease, *Spe*I (5'-ACTAGT-3'; pattern designation S). Although fewer bands were seen compared to PFGE, ribotyping of these isolates using *Spe*I-digested genomic DNA largely confirmed the PFGE results in that the sporadic isolates gave unique profiles and only three closely related ribotype profiles were detected among the outbreak isolates. Two Malaysian isolates of *S. paratyphi* A included for comparison gave patterns very different from the Indian isolates by both PFGE (F = 0.44-0.65) and ribotyping. Also, it was determined that isolates A-117 (X1/S1) and A-123 (X2/S2) belonged to the index cases and that, as the outbreak progressed, other patterns (X3/S3 and X4/S4), which differed from the original patterns by one to four bands, appeared during weeks 2 to 3 of the outbreak. Notably, patterns X1 and X2 reappeared at the end of the outbreak.

Although molecular analysis of *S. typhi* and *S. paratyphi* B by ribotyping (2,4) and PFGE (3) has been reported, to the best of our knowledge the present study is the first performed with *S. paratyphi* A. The data obtained agree with those observed for *S. typhi* (3) in that outbreak isolates are more clonal and limited in diversity, whereas sporadic isolates are more diverse genetically and belong to unrelated clones. According to the criteria of Tenover et al. (5), it seems likely that the present outbreak was associated with two distinct clones/strains of *S. paratyphi* A (X1/S1 and X2/S2) that are related (5) but have distinct PFGE profiles. This observation is perhaps not surprising given the fact that both clones are phage type 1 and that contaminated potable water was incriminated in the outbreak (1). The PFGE results were largely confirmed by ribotyping, although this technique appears to be slightly less sensitive and discriminating in that fewer bands were seen and the differences between outbreak isolates were much less obvious.

We thus conclude that the outbreak in New Delhi, India, was caused by two related but distinct clones of *S. paratyphi* A. There also appears to be substantial genetic diversity among *S. paratyphi* A strains as the Malaysian isolates were very different from those from India. The data also suggested minor genetic changes among the *S. paratyphi* A isolates during the 2-month outbreak. This observation agrees with the high mutation rates noted among pathogenic *Salmonella* spp. (6) and the plasticity of the genome of salmonellae associated with enteric fever (7). How these changes affected the biologic behavior of these isolates will be the subject of further study. Our study reaffirms the usefulness of PFGE and ribotyping in the molecular typing and discrimination of individual *Salmonella* isolates for epidemiologic investigations.

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Unrecognized Ebola Hemorrhagic Fever at Mosango Hospital during the 1995 Epidemic in Kikwit, Democratic Republic of the Congo

To the Editor: We report here the clinical description of a hemorrhagic syndrome observed